

The reduced motility of mitochondria lacking Puf3p causes them to fragment (middle) or aggregate (right).

Meting out mitochondria

Cells with fewer mitochondria must be careful to dole the organelles out equally to their progeny. As Garcia-Rodriguez et al. show on page 197, Puf3p reduces mitochondrial numbers when demand for the organelles is low but also ensures that the few remaining mitochondria are well connected to the machinery that pushes them around during reproduction.

Researchers knew that Puf3p latches onto messenger RNAs that encode mitochondrial proteins, and then accelerates their breakdown. A related protein, Puf1p, helps sort mitochondria when a yeast cell buds to produce offspring. Puf1p links mitochondria to the Arp2/3 complex, which generates the force to propel the organelles into the new bud.

Garcia-Rodriguez et al. tested whether Puf3p does likewise. The team found that Puf3p fastens Arp2/3 to the mitochore, a complex of integral mitochondrial membrane proteins that is required for mitochondrial movement. The connection is essential for shuttling mitochondria into the bud. In cells lacking Puf3p, mitochondria bunch up, break apart, and move sluggishly.

The scientists also found evidence that Puf3p impedes the formation, or biogenesis, of new mitochondria. When yeast were switched from a sugar diet to eating ethanol, they cranked out extra mitochondria and slashed production of Puf3p, suggesting that the protein was previously standing in the way of making new organelles. Furthermore, the shifted cells were sickly if they were forced to overexpress Puf3p. Garcia-Rodriguez et al. propose that cells use Puf3p to reduce numbers when the mitochondria are not needed, but also to ensure that the reduction does not lead to mitochondrial loss and cell death. **JCB**

Thrown for a D-loop

I won't force James Watson to retitl the *Double Helix*, but new research suggests that the three-stranded stretches that frequently turn up in mitochondrial DNA (mtDNA) aren't junk. Instead, the triple-stranded forms might provide a scaffold for a protein that helps mitochondria organize their DNA, as He et al. show on page 141.

Researchers have known for more than 30 years that one section of mtDNA often carries an additional strand, creating a displacement loop, or D-loop. However, the conventional wisdom held that D-loops were nonfunctional leftovers of incomplete replication. He et al. began to doubt that explanation after "we found a protein that is in love with D-loops," says team leader Ian Holt. Other researchers had previously identified the protein, ATAD3, but knew nothing about its job in mitochondria.

The researchers discovered that ATAD3 prefers to latch onto D-loops, even in solutions where double-stranded DNA is 1,000 times more abundant. ATAD3 also helps stabilize complexes with multiple copies of mtDNA. When the scientists tracked ATAD3 down in the mitochondria, they learned that it often hangs out in the clusters of mtDNA called nucleoids. ATAD3 may either help mtDNA copies segregate from each other or cluster multiple copies of them into nucleoids. Herding mtDNA into nucleoids might offer the strands protection from the reactive oxygen species that are prevalent in mitochondria or allow the cell to control the number of mtDNA copies. **JCB**

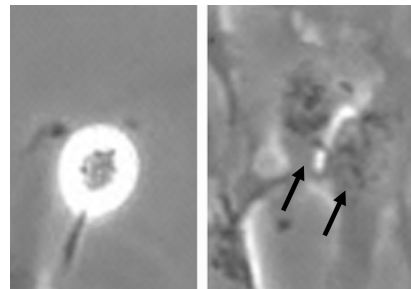
Cycling on without centrosomes

Centrosomes are dynamic leaders that propel the cell cycle forward. Or are they followers swept along by change? On page 173, Uetake et al. offer evidence that reconciles these disparate views. The group demonstrates that, although centrosomes are not required for cell cycle progression, their loss halts the process under stressful conditions.

Researchers long thought that centrosomes were passive participants in the cell cycle. But several recent studies found that removing or injuring centrosomes prevents cells from entering S phase, suggesting that the centrosome isn't just along for the ride.

To resolve this uncertainty, Uetake et al. excised the centrosomes from two kinds of normal cells. The cells nicely rolled through G1 into S phase, suggesting that centrosomes are not normally vital for entering S phase.

To explain why previous reports found differently, the authors considered stressed cells, which often stall in G1. They proposed that centrosome loss might put cells under small amounts of pressure that create stalls when added to other stresses.



A cell lacking centrosomes (left) can still undergo mitosis (right).

Uetake et al. tested the idea by removing centrosomes from cells and then exposing them to blue light stress. Neither stimulus alone halted the cell cycle, but the combination did. Cells exposed to both stresses could advance through G1 if the researchers first blocked p38, which switches on a G1 arrest when cells are under duress. **JCB**